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RESEARCH

Long-term survival following traumatic brain injury

GRAHAM RATCLIFF[1], ANGELA COLANTONIO[2], MICHAEL ESCOBAR[3], SUSAN CHASE[4], & LEE VERNICH[3]

[1]*HealthSouth Harmarville Rehabilitation Hospital, Pittsburgh, PA, USA,* [2]*Department of Occupational Therapy, University of Toronto, Canada,* [3]*Department of Public Health Sciences, University of Toronto, Canada, and* [4]*Working Order, Pittsburgh, PA, USA*

Abstract

Purpose. The study used a retrospective cohort design to establish long-term mortality rates and predictors of mortality for persons after moderate to severe traumatic brain injury (TBI).

Method. Consecutive records of persons with moderate to severe TBI who were discharged from a large rehabilitation hospital in Pittsburgh, Pennsylvania in the years 1974 – 1984, 1988 and 1989 were reviewed.

Results. Six hundred and forty-two eligible individuals were identified and mortality was ascertained up to 24 years post injury. One hundred and twenty-eight of these individuals were found to be deceased. Poisson regression analyses revealed at least a 2-fold increased risk for mortality compared to the general population. Pre-injury characteristics and levels of disability at discharge from in-patient rehabilitation were among the strongest predictors of mortality.

Conclusions. These data constitute evidence for premature death in the post-acute TBI population following a moderate to severe head injury and are discussed in relation to other research in the area.

Keywords: *Traumatic brain injury, mortality, standardized mortality ratio, death rate, long-term survival*

Introduction

Traumatic brain injury (TBI) is a leading cause of death and disability, particularly in young adult males [1] and a significant issue for rehabilitation professionals [2]. Published estimates of head injury-associated death rates range from a conservative estimate of 15 per 100 000 to 30 per 100 000 [3]. The differences are probably attributable to different methodologies and the trend towards fewer fatalities, particularly from motor vehicle accidents, since 1980 [4 – 6]. However, most of the deaths documented in these studies occurred in the acute phase following the accident. Recent studies that include late mortality have typically tracked this retrospectively by surveying death certificates and counting the number of deaths attributable to head injury [3 – 6]. These studies have followed the clear inclusion criteria for head injury-associated death (HIAD) established by Sosin et al. [7] in requiring both that head injury be listed in the sequence of conditions that resulted in death and that an injury condition be coded as the underlying cause of death.

While these criteria have contributed to reliable estimates of mortality, they may underestimate the number of late deaths in which the head injury is neither the immediate, proximate cause of death nor the primary condition for which a patient is being attended at the time of death. Max *et al*. [3], for example, found only 785 deaths from the late effects of head injuries experienced earlier among a group of 36 712 fatalities, although the few prospective studies that address the issue and individual case reports would have led one to expect higher figures. Thus, Strauss *et al.* [8] report continuing excess mortality in children more than 6 months after injury. Baguley *et al.* [9] found a

Correspondence: Dr G. Ratcliff, Neuropsychology HealthSouth Harmarville Rehabilitation Hospital, P.O. Box 11460 Guys Run Road, Pittsburgh, PA, USA. Tel: 1 412 826 2783. Fax: 1 412 828 7931.

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significantly greater mortality rate at a follow-up of 8 months to 11 years in individuals treated for traumatic brain injury in an Australian rehabilitation hospital, compared with that expected on the basis of national life table data. In two older series reported from England, Roberts [10] noted an increased death rate at follow up in younger patients and Fahy *et al.* [11] reported an estimated 6-fold increase in deaths after discharge from hospital in a less formal follow-up study. Certainly, these prospective studies include deaths which were not attributable to head injury and acknowledge the difficulty in adjudicating those to which head injury made a contribution [10]. They also tend to be older and not all prospective studies have found increased mortality rates [12].

A recent study by Shavelle *et al.* [13] avoids some of these problems. They tracked individuals who had received services from the California Department of Developmental Services because of the appearance of cognitive impairment attributed to either a motor vehicle accident or cranial injury and who had survived for at least 1 year after the injury. Mortality was established by reference to computer records of death certificates but the authors counted all deaths, not merely those attributed to head trauma. They found an increased death rate for all causes with a standardized mortality rate of 3.1 following TBI compared with the general population after adjusting for age and gender, particularly in the first 5 years after injury. However, as they point out, their subjects were all receiving state services because of long-term cognitive or communication disorders and probably represent a much more disabled group than would be obtained in most hospital series.

It is not clear, therefore, to what extent a history of head injury constitutes a risk factor for early death in adults who survive the acute phase. Factors found to influence long-term survival include basic functional skills such as mobility and self-feeding [8,9,13], post-traumatic epilepsy in veterans [14] and more direct measures of the type and severity of injury [10]. Known effects of brain injuries such as cognitive changes, risk taking behavior and mood disorder have also been cited as potential factors leading to increased risk of death, though their role has not been established in formal studies.

We now report data from a study of long-term outcome after moderate to severe head injury that has been described previously in this journal [15]. The aim of this paper is to document the impact of a history of head injury on long-term survival in a population of persons who received inpatient rehabilitation for moderate to severe head injury. In addition, we document predictors of post-acute mortality in this population. Predictions based on factors present at time of discharge are particularly relevant to healthcare providers who must estimate prognosis [16].

Methods

Subjects

The study utilized a retrospective cohort design. A cohort of subjects injured 8–24 years previously were recruited from the medical records of a large, 200 bed comprehensive rehabilitation treatment facility in Pennsylvania. This facility serves a mixed urban and rural region with referrals from community hospitals, as well as tertiary care centers. It was the main provider of rehabilitation services for persons with head injury in the area during most of the relevant period. Patients with head injury were first identified from medical records by reference to specific ICD-8 and ICD-9 codes for head injury (800–801.9; 803–804.9; 850–854.9) that include skull fracture and intracranial injuries [17]. The range includes concussion; cerebral laceration and contusion; subarachnoid, subdural, and extradural hemorrhage following injury; other unspecified intracranial hemorrhage following injury; and intracranial injury of other and unspecified nature. All records that had head injury ICD codes were reviewed for accuracy of coding and severity of head injury. Individuals who had non-traumatic injuries or who had a co-morbid spinal cord injury in addition to head trauma were excluded from the sample since they represent a distinctly different group. Participants were all at least 14 years of age at time of injury as the rehabilitation hospital through which they were selected did not routinely admit patients below this age.

We examined all consecutive records of former patients discharged during the years 1974–1984. In addition, we selected 2 years from a more recent time period, 1988 and 1989. We excluded the years 1985–1987 purely on practical grounds in order to limit the total sample to a number that we could reasonably expect to visit during the study period and we reviewed records back to 1974 in order to maximize the potential length of follow-up. This record review resulted in 642 eligible participants. Of these, 128 persons were found to be deceased. There were 42 persons that we were not able to trace, but we assumed that they were not deceased since our review of mortality databases did not reveal them as such.

Data collection procedure

Baseline information about study participants was abstracted from medical records. Study variables were classified according to the following cate-

gories: (1) pre-injury demographics, (2) pre-injury social characteristics, (3) severity of injury, (4) functional status at discharge, and (5) mortality outcome.

Pre-injury demographic characteristics. Information about gender, race, education, marital status and living arrangements at time of injury was abstracted from medical records.

Pre-injury social characteristics. The medical record was reviewed for prior history of (a) problems with alcohol and substance abuse, (b) a criminal record, (c) psychiatric disorders and (d) personal/social problems, such as homelessness, or recent death of a spouse. Any mention of these problems was noted. We also created a summary variable coding the presence of any of these conditions.

Injury severity variables. In this category we recorded (a) date of injury, (b) mechanism of injury, and (c) ICD-8/ICD-9 codes.

Other information relating to the nature and severity of the injury was taken from the *History and Physical* and *Physical Medicine and Rehabilitation Evaluation,* conducted by the admitting physicians in the rehabilitation hospital. Although acute hospital records were no longer maintained as part of the rehabilitation record 8 – 24 years after the patient's discharge, rehabilitation charts were typically quite detailed and included an abstract of the acute hospital records. However, in the older records, sophisticated imaging data such as CT or MRI would not routinely have been available and GCS scores had frequently not been computed, particularly when the acute hospital from which the patient had been admitted was not a trauma center. Accordingly, estimates of the durations of coma and PTA were derived from the admitting physician's account of the acute care course. However, since we did not have complete information from acute care records, we did not include these estimates in the analysis. Note was also made of any mention of a range of neurological data relating to type and location of lesion, history of neurosurgical intervention, neurological signs and symptoms, hypoxia, hypotension and information on pupil size and reactivity.

An injury severity score was computed by using ICD-8/ICD-9 codes at discharge as has been done by other investigators [18,19]. As this was a continuous measure ranging from 0 to 75, intervals were developed for purposes of analyses and are shown in Table III

Functional status. Functional status was assessed by reference to level of independence in certain activities of daily living (ADLs) upon discharge from the rehabilitation hospital. We recorded the ability of clients to bathe, groom, dress, eat, get from bed to chair, use the toilet and walk across the room at the time of discharge. Independence in these activities was recorded as done (a) without help, (b) with some help, and (c) unable to do. This information was routinely available in Occupational Therapy notes and discharge summaries. We also created a variable that totaled the number of limitations in all these areas.

Mortality

Mortality was ascertained by using a tracer service, and/or by confirmation of death by relatives. Public databases such as the Social Security Index were consulted to obtain dates of death. We were able to retrieve year of death for all but one person who was subsequently excluded from the analysis. Also excluded was one person who died on the same day as discharge. Therefore, our analyses were based on a sample of 640. Death Certificates were obtained for persons who died in Pennsylvania, the majority of the sample. The study protocol was approved by the Internal Review Boards of both the University of Toronto and the rehabilitation hospital where participants were recruited.

Statistical analysis

The main method of analysis used Poisson regression [20] to model the relative risk of death as compared to the expected death rates calculated from published mortality rates in the Pennsylvania Vital Statistics Table [21]. We calculated each person's expected probability of death during the time of exposure using standard methods to calculate incident rates [22]. The time of exposure to death is an interval defined to begin when the study participant was discharged from the rehabilitation facility and end at the time of death or, if the person had not died, at the conclusion of data collection in 1997. Using a Poisson regression we could estimate the ratio of the observed number of deaths to the expected number of deaths. This results in the standardized mortality ratio (SMR) which constitutes a measure of the increased mortality rate of the cohort. We were also able to examine which variables affected the SMR.

In order to calculate the probability of a subject dying during the time that the subject was exposed to death, the instantaneous rate function, also called the hazard function, was calculated. The published mortality rates for Pennsylvania were used to implicitly define the instantaneous rate function as a step function. From the mortality

tables, we obtained for each age group A and calendar year B the rate of deaths per year, which we will call r (A, B). Therefore, the instantaneous rate function at time t is equal to r (A, B) for a subject if time t is in calendar year B and if the subject is in age group A at time t. Next, the cumulative incident rate function is calculated. The cumulative incident function is the integral of the instantaneous death rate over the exposure period. Since the instantaneous death rate is a step function, then the cumulative incident rate function is equal to the weighted sum of the rates r (A, B) for a subject with weights equal to the percentage of the calendar year B that the subject is both in age group A and exposed to death. One can then calculate the probability of surviving since the survival probability is equal to the negative of the logarithm of the cumulative incident function. The probability of dying can now be obtained since it is equal to one minus the probability of surviving.

Each subject could then be modeled by a Bernoulli random variable, so the sum of many subjects could be modeled as a Poisson random variable allowing us to use a Poisson regression model with a logarithm link function for the analysis. In the Poisson regression, the logarithm of the expected number of deaths is used as the offset term. The Poisson regression enables one to test for differential death rates across the levels of a categorical variable. The intercept term in the model is the logarithm of the relative risk of death in the head injury cohort as compared to the overall population, which is also the logarithm of the SMR. If this term is significantly different than zero, it would signify that the cohort has a higher risk of death than the general population. A likelihood ratio test was used to determine if the intercept term was significant in the model. This was done by calculating the model with no independent variable and with and without the intercept term. A significant effect term in the model tests to see if the relative risk is different for different levels of the effect being tested. The SAS computer program Proc GENMMOD was used to carry out the calculations in this paper [23].

We also attempted to model mortality outcome. To this end, variables were first examined for outliers and skewness and were transformed where necessary. Modeling of the mortality outcome began with a bivariate Poisson to see which of the predicted variables of interest were statistically significant, bearing in mind that there are often many variables that measure the same basic underlying concept. Because of the large number of variables with only a few being significant in the basic bivariate analyses, only the significant and near significant were examined in the subsequent multi bivariate analysis. In this analysis, we initially performed a forward stepwise regression to see which variable entered in the model. Also we performed an enhanced stepwise regression where, instead of entering the 'best' variable, we would enter the variables which were 'near best', thereby assembling a collection of good models. Typically, these models contained similar variables to the 'best' subset model.

Results

The demographics of the sample are shown in Table I. As expected in this population, males outnumbered females by nearly 3 – 1. The majority were young adults at the time of injury (mean age was 34.8) though a substantial minority were aged over sixty (12.8%). Mean educational level was 11.6 years with a median of high school (12 years), an educational level of less than high school sometimes being attributable to the fact that the injury occurred before the individual had graduated. Thirty-six percent were married or living with a significant other at the time of injury, whereas 47.5% had never been married.

Mechanism of injury is also shown in Table I. The majority were transportation related (66.6%), predominantly involving automobile crashes. Falls were the next most frequent cause (16.4%). There was a relatively low rate of injuries related to interpersonal violence including only 10 (1.6%) gunshot wounds.

Table I also presents observed and expected deaths by age at time of injury, together with the number of persons in each age group and the relative risk ratio. We find that 19.7% of the sample had died between discharge from the rehabilitation hospital and the date of follow-up, a rate over two and a half times greater than that expected on the basis of Pennsylvania data (SMR = 2.78, $\chi^2 = 96.35$, df = 1, $P < 0.0001$). Note that the SMR is consistently elevated for all levels of all the variables in Table I, but that there is no significant difference in SMR over the level of these variables in bivariate analyses as determined by likelihood ratio tests.

For example, for the age categories, the SMR ranges from 1.60 to 3.58. Since the chi-square value is 6.53 on 7 degrees of freedom with an associated P value of 0.4798, then we would conclude that there is no significant difference between the levels of age. Likewise, there is no significant difference in SMR over the levels of the demographic variables of gender, pre-injury education, marital status or mechanism of injury in bivariate analyses.

Conversely, Table II shows that a documented history of prior problems with alcohol abuse, substance abuse, other personal/social problems, and the composite measure of social/behavioral problems were all significant modifiers of the

Table I. Demographic characteristics of the sample.

	%	*N*	Observed no. of deaths	Expected no. of deaths	Estimated SMR	Test of differences between levels of the variable
Sex						
Male	72.50	464	87	28.71	3.03	$\chi^2 = 1.8$
Female	27.50	176	39	16.60	2.35	df = 1
Total:		640	126	45.31	2.78	$P = 0.1802$
Age at Injury						
14 – 19	19.4	124	7	2.06	3.40	$\chi^2 = 6.53$
20 – 24	19.5	125	4	2.50	1.60	df = 7
25 – 29	16.3	104	7	2.01	3.48	$P = 0.4798$
30 – 39	13.6	87	10	2.83	3.53	
40 – 49	10.3	66	17	4.32	3.94	
50 – 59	8.1	52	20	7.50	2.67	
60 – 69	6.3	40	28	9.19	3.05	
70 +	6.6	42	33	14.90	2.21	
Total:	100.0	640	126	45.31	2.78	
Education						
No High School diploma	33.2	208	56	17.70	3.16	$\chi^2 = 2.14$
High School Diploma	42.7	268	39	14.46	2.70	df = 3
Some College	14.8	93	9	3.78	2.38	$P = 0.5434$
4 or more years college	9.3	58	12	5.67	2.12	
*Total:		627	116	41.61		
Marital status at injury						
Never married	47.48	301	22	7.61	2.89	$\chi^2 = 5.7$
Married or living w/someone	36.12	229	60	24.97	2.40	df = 3
Divorced/separated	12.46	79	22	4.91	4.48	$P = 0.1270$
Widowed	3.94	25	19	7.07	2.69	
*Total:		634	123	44.54	2.76	
Race						
Nonwhite	4.69	30	8	2.14	3.74	$\chi^2 = 0.7261$
White	95.31	610	118	43.17	2.73	df = 1
Total:		640	126	45.31	2.78	$P = 0.3942$
Mechanism of injury						
MVA	66.56	426	46	20.65	2.23	$\chi^2 = 4.7$
Fall	16.41	105	52	16.91	3.08	df = 2
Other	17.03	109	28	7.76	3.61	$P = 0.0955$
Total		640	126	45.31	2.78	

*Observations with values of 'not known' were not included in the analysis.

mortality rate. For example, for the composite measure, the SMR for no problems reported was 2.05 but if at least one of the problems was noted the SMR jumped to 5.82 ($\chi^2 = 29.71$, df = 1, $P < 0.0001$).

Neither of the injury severity measures showed a significant effect on mortality (Table III). However, Table IV shows that the patient's functional status on discharge, which may itself be related to injury severity, was significant for all variables. The strongest indicators were difficulties with grooming and eating. The summation variable also appeared quite strong.

Table V indicates that the year of follow-up is not a significant modifier ($\chi^2 = 26.94$, df = 18, $P = 0.0802$) of the mortality rate. That is, there is not a significant preponderance of deaths in the early part of the follow-up period and increased death rates are present throughout the 19 + years of follow-up. Although the SMR for the first year of follow-up appears to be high (SMR = 4.95), the SMR for year 7 is higher (5.47). Therefore, we do not have any significant evidence of a first year increase of mortality in our data.

Table VI contains the results of the multivariate analyses. A stepwise regression would choose model 1 as the primary model. Both models contain the variable for the sum of behavior problems. Then, either the variable for grooming or eating is entered, since they have almost identical chi-squared values ($\chi^2 =$ 14.1974 and $\chi^2 = 14.1614$, respectively). In model 1, if someone has no prior, documented behavioral disturbances/problems and no problem with grooming, there is an increased mortality ratio of 1.69. If any prior behavioral/social problems are noted, then this risk is further multiplied by 2.98. The mortality risk is multiplied by 1.31 if there is some problem with grooming. The multiplier is 2.99

Table II. Pre-injury social/behavioural problems.

	%	*N*	Observed no. of deaths	Expected no. of deaths	Estimated SMR	Test of differences between levels of the variable
Alcohol abuse						
No/not mentioned	81.88	524	88	39.08	2.25	$\chi^2 = 22.56$
Yes	18.13	116	38	6.23	6.10	df = 1
Total		640	126	45.31		$P = 0.0001$
Substance abuse						
No/not mentioned	95.43	605	120	44.56	2.69	$\chi^2 = 4.93$
Yes	5.47	35	6	0.75	8.00	df = 1
Total		640	126	45.31		$P = 0.0264$
Criminal record						
No/not mentioned	98.28	629	126	45.1	2.79	$\chi^2 = 1.16$
Yes	1.72	11	0	0.21	0.00	df = 1
Total		640	126	45.31		$P = 0.2825$
Psychiatric disorder						
No/not mentioned	95.16	609	121	44.21	2.74	$\chi^2 = 1.08$
Yes	4.84	31	5	1.1	4.55	df = 1
Total		640	126	45.31		$P = 0.2987$
Other personal/social problems						
No/not mentioned	86.25	552	103	42.04	2.45	$\chi^2 = 16.56$
Yes	13.75	88	23	3.27	7.03	df = 1
Total		640	126	45.31		$P = 0.0001$
Any of the above five problems						
No/not mentioned	71.41	457	75	36.54	2.05	$\chi^2 = 29.71$
Yes	28.59	183	51	8.77	5.82	df = 1
Total		640	126	45.31		$P = 0.0001$

Table III. Injury severity measures.

	%	*N*	Observed no. of deaths	Expected no. of deaths	Estimated SMR	Test of differences between levels of the variable
Pupil dilation						
None or not noted	53.52	342	74	29.68	2.49	$\chi^2 = 2.49$
1 or 2 eyes	46.48	297	52	15.63	3.33	df = 1
Total		639	126	45.31	2.78	$P = 0.1144$
ISSINT Illness						
Severity Score*						
Level 1 (4–13)	11.22	68	21	5.04	4.16	$\chi^2 = 7.91$
Level 2 (14–22)	21.78	132	46	18.36	2.51	df = 4
Level 3 (24–27)	28.22	171	30	8.74	3.43	$P = 0.0950$
Level 4 (29–34)	25.91	157	14	7.33	1.91	
Level 5 (35–54)	12.87	78	6	3.11	1.93	
Total:		606	117	42.58	2.75	

*This variable transformed a scale with a range of 0–75 into the categories above to normalize the data.

if individuals are completely incapable of grooming themselves.

Two other strong models included the same covariates but substituted alcohol abuse or social problems for the composite variable denoting the sum of behavioral problems. The multipliers were very similar for these alternate models, but the models including social/behavioral problems were predominant. Therefore, these models were chosen and are reported here.

Discussion

Our findings show that there is more than a 2-fold increase in long-term mortality for this group of individuals with moderate to severe head injury as compared to the general population in Pennsylvania. The risk is more pronounced in the most disabled groups as indicated by level of independence in basic ADLs. It should be emphasized that these deaths all occurred after discharge from a rehabilitation facility

Table IV. Functional status at discharge: activities of daily living.

	%	*N*	Observed no. of deaths	Expected no. of deaths	Estimated SMR	Test of differences between levels of the variable
Bathing						
No Problem	70.60	449	57	24.67	2.31	$\chi^2 = 9.2145$
Some problem	21.07	134	44	15.76	2.79	df = 2
Yes	8.33	53	24	4.74	5.06	$P = 0.01$
Total:		636	125	45.17		
Grooming						
No Problem	81.45	518	77	32.04	2.40	$\chi^2 = 12.21$
Some problem	12.89	82	30	10.44	2.87	df = 2
Yes	5.66	36	18	2.7	6.67	$P = 0.0022$
Total		636	125	45.18		
Dressing						
No Problem	70.91	451	59	23.97	2.46	$\chi^2 = 9.1$
Some problem	21.07	134	43	16.88	2.55	df = 2
Yes	8.02	51	23	4.33	5.31	$P = 0.0106$
Total		636	125	45.18		
Eating						
No Problem	79.40	505	76	32.97	2.31	$\chi^2 = 11.83$
Some problem	14.78	94	31	9.12	3.40	df = 2
Yes	5.82	37	18	3.09	5.83	$P = 0.0027$
Total		636	125	45.18		
Bed to chair						
No Problem	67.45	429	52	21.89	2.38	$\chi^2 = 6.7$
Some problem	25.31	161	52	18.93	2.75	df = 2
Yes	7.23	46	21	4.36	4.82	$P = 0.035$
Total		636	125	45.18		
Toilet use						
No Problem	77.01	489	66	27.7	2.38	$\chi^2 = 8.18$
Some problem	14.02	89	34	12.31	2.76	df = 2
Yes	8.98	57	25	5.15	4.85	$P = 0.0167$
Total		635	125	45.16		
Walking across room						
No Problem	69.50	442	57	23.3	2.45	$\chi^2 = 7.95$
Some problem	21.54	137	44	17.04	2.58	df = 2
Yes	8.96	57	24	4.83	4.97	$P = 0.0188$
Total		636	125	45.17		
Summation of activity limitations						
0	55.75	354	36	16.89	2.13	$\chi^2 = 11.72$
1 – 7	33.86	215	60	21.99	2.73	df = 3
8 – 13	5.98	38	15	4.15	3.61	$P = 0.0084$
14	4.41	28	14	2.12	6.59	
Total		635	125	45.16	2.77	

to which patients had been transferred following a prior acute hospital stay. Deaths were found in all age groups and throughout the length of the follow up period. There was no tendency for them to cluster in the years immediately following discharge from hospital, unlike the predominance of deaths in the first 5 years reported by Shavelle *et al.* [13].

As Roberts [10] has pointed out, it is often difficult to determine with certainty whether a prior head injury was a contributing cause to an individual's death, even when the reported cause of death is known. Certainly, many of the deaths in our series could not meet the criteria for HIAD defined by Sosin *et al.* [7]. The fact that pre-injury behavioral characteristics such as alcohol abuse were strong predictors suggests that some characteristic of the population experiencing head injury, rather than head injury *per se* contributes to the increased death rate. A propensity for risk-taking behavior and substance abuse would be obvious potential candidates. A recent study has found that these factors predict post acute death in a trauma population overall [24], though this does not seem to have been the case in Shavelle *et al.*'s [13] group.

On the other hand, there is evidence that population factors present prior to the injury are not the sole explanation. Thus, increased death rates were also observed in individuals without known pre-morbid

Table V. Standardized mortality ratio (SMR) for years of follow-up.

Year of Follow-up	% Remaining	No. of persons each year	Observed no. of deaths	Expected no. of deaths	Estimated SMR	Test of differences between levels of the variable
1	100.00	639	11	2.221	4.95	$\chi^2 = 26.94$
2	98.28	628	10	4.485	2.23	df = 18
3	96.56	617	12	4.357	2.75	$P = 0.0802$
4	94.52	604	8	4.030	1.99	
5	93.27	596	6	3.748	1.60	
6	92.33	590	3	3.657	0.82	
7	91.86	587	19	3.475	5.47	
8	88.89	568	11	3.120	3.53	
9	87.17	557	7	2.881	2.43	
10	69.95	447	5	2.177	2.30	
11	52.90	338	4	1.521	2.63	
12	52.11	333	5	1.454	3.44	
13	51.33	328	6	1.459	4.11	
14	50.39	322	1	1.458	0.69	
15	41.78	267	4	1.280	3.13	
16	33.80	216	2	0.979	2.04	
17	25.82	165	1	0.676	1.48	
18	20.66	132	2	0.539	3.71	
19 +	xxxx	xxxx	3	1.298	2.31	

Table VI. Multivariate logistic regression: final models.

	Level	RR	Lower CI	Upper CI	Chi-square	DF	*P* value
MODEL 1							
Intercept		1.69	1.29	2.23			
Any pre-injury behavioural problem*	No	REF			30.1974	1	0.0001
	Yes	2.98	2.09	4.27			
Grooming	No problem	REF			14.1974	2	0.0008
	Somewhat	1.31	0.86	2.00			
	Yes	2.99	1.79	5.00			
MODEL 2							
Intercept		1.66	1.26	2.17			
Any pre-injury behavioural problem*	No	REF			30.1974	1	0.0001
	Yes	3.01	2.10	4.32			
Eating	No problem	REF			14.1614	1	0.0008
	Somewhat	1.42	0.94	2.16			
	Yes	2.90	1.73	4.87			

*Any pre-injury behavioral problem includes alcohol abuse, substance abuse, a criminal record, psychiatric disorders or personal/social problems.

substance abuse or other behavioral problems. In addition, functional limitations noted at the time of discharge from the rehabilitation hospital and presumably caused by the injury were also predictive. Our finding that limitation in eating was one of the most important predictors in the multivariate analyses was also found by Straus *et al.* [8] in a younger rehabilitation population and a composite functional independence measure was a strong predictor of mortality at shorter term follow-up in Baguley *et al.*'s [9] Australian series. Injury severity variables did not play a great role in predicting mortality in our data. This could be due to the fact that our use of retrospective data from old medical records limited our ability to access more sophisticated measures. For example, MRI was unknown when our earliest participants were injured, CT was not standard and GCS scores were not reported as frequently as would now be the case. In addition, length of unconsciousness and PTA were not always reported, or were reported in only general terms that were not suitable for analysis.

Whatever the mechanism underlying the effect, our data indicate that a history of head injury constitutes a risk factor for mortality, even in individuals who survive through the rehabilitation

phase and have not been selected on the basis of evident, persistent disability. This adds to our knowledge about the long-term outcome from head injury and, in our opinion, the methodology adopted here is an important complement to the retrospective studies of HIAD using death certificates and associated databases. We can be sure of including deaths that may have been the indirect result of head injury even when no such attribution was made on the death certificate. An analysis of the reported causes of death in our sample will be the subject of a separate paper but we are already aware of some such cases. Potential examples include deaths from pneumonia in individuals in long-term care and accidental deaths in individuals who are likely to have had severe cognitive deficits. Baguley *et al*. [9] speculate that inactivity-related morbidity may have been a factor in the increased death rate, predominantly from cardiorespiratory arrest and broncho pneumonia/sepsis in their study. We suspect that such indirect effects of head injury may contribute to more than the 5.3% of deaths in which head injury was recorded as contributing to death but not related to the underlying cause of death on the death certificates reviewed by Sosin *et al.* [7].

There are several limitations to our study. As noted previously, the inherently poor quality of our data related to injury severity variables such as GCS severely limits our ability to comment on the predictive power of variables in this category. In addition, our measures of pre-injury characteristics were limited to data from medical records which may not have been collected in a systematic way. Our earliest participants were injured as long ago as 1973 and life expectancy following head injury may have changed since then. Improved management may have led to better outcomes or, conversely, improvements in acute care may have led to the survival of more severely injured individuals who may or may not be more likely to succumb in the long term. Our population had all received inpatient rehabilitation services and may be biased towards more severe cases compared with the whole population of acute hospital discharges although it includes individuals whose injuries did not cause disabilities that would have made them eligible for inclusion in the Shavelle *et al*. [13] series. Nevertheless, the overall SMR of 2.78 in our sample is very close to their value of 2.7 for deaths occurring more than 5 years post injury. Our subjects also came from a restricted geographic region, and the results may not generalize to other populations.

The demographics of our population are, however, quite similar to those of other community based studies of head injury in which there is a predominance of young men and where motor vehicle accidents are the most common mechanism of injury. Recruiting from a rehabilitation hospital gave us access to in-depth information relevant to post acute status such as activity limitations that would not be systematically available in acute care records. The contribution of improved acute management to the reduction in fatalities from TBI since the beginning of our study period in 1974 is probably chiefly attributable to the prevention of early deaths which would have occurred before subjects became eligible for inclusion in our study rather than the late mortality studied here. We were able to survey a continuous series of discharges and the stability of the population in western Pennsylvania was a factor in our ability to trace 93% of potentially eligible participants. The rehabilitation facility from which patients were recruited treated a large percentage of persons on medical assistance and many of the injuries occurred in motor vehicle accidents at a time when no fault insurance typically paid for rehabilitation. Patients came to rehabilitation from a number of different acute care settings including community hospitals and tertiary care trauma centers in a mixed rural and urban region. We doubt, therefore, that the sample is substantially biased due to differential access to rehabilitation services. In addition, within our sample, there was no differential effect of socio-economic status.

Several issues are unresolved and point to the need for more research with respect to factors influencing mortality and morbidity in this population. This should include further detailed analysis of the causes of death in individuals with a history of head injury, including those without severe, persisting disability as well as counts of the number of deaths attributed to head injury in the population. The distribution of causes of death long after head injury should be compared with that found in other high-risk populations matching for age, sex and preferably, other demographic variables. Apolipoprotein E genotype may also be relevant [25]. Studies that take account of these variables would provide information for health care providers and social service agencies interested in the prognosis of individuals who have survived the acute phase after head injury. They might also suggest strategies that could be put into place to reduce the risk of premature death, particularly in higher risk persons as identified in this paper.

In addition, this study highlights the importance of obtaining a good pre-injury history and a functional assessment that includes activities of daily living after injury.

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